Journal of Cardiology Cases 12 (2015) 16–19

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Stress-induced cardiomyopathy accompanied by heat stroke

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ARTICLE INFO

Article history:
Received 8 December 2014
Received in revised form 10 March 2015
Accepted 25 March 2015

Keywords:
Stress-induced cardiomyopathy
Takotubo-cardiomyopathy
Heat stroke
Ventricular tachycardia
Aberrant conduction

ABSTRACT

Heat stroke is a severe illness characterized by a core temperature >40 °C (104 °F) and central nervous system abnormalities, such as delirium, convulsions, or coma, resulting from exposure to environmental heat or strenuous physical experience. We describe a case with high body temperature and coma, who also presented with wide QRS tachycardia and shock. He was initially treated for heat stroke and wide QRS tachycardia. Serial electrocardiographic change and echocardiographic findings suggested this was complicated with stress-induced cardiomyopathy. This case demonstrates the importance of recognizing that serious myocardial damage can result from heat stroke.

<**Learning objective:** Heat stroke can cause multiple organ failure and the presentation of circulatory failure in heat stroke may be the sign of myocardial dysfunction. To distinguish acute coronary syndrome and stress-induced cardiomyopathy, both of which could be evoked by heat stroke, the evaluation of coronary artery is necessary. Stress-induced cardiomyopathy may cause lethal arrhythmia or circulatory collapse in acute phase. Invasive circulatory monitoring is recommended in patients with heat stroke.>

Introduction

Heat stroke is a medical emergency commonly encountered in small children and socioeconomically deprived elderly people, but can also occur in healthy young persons after strenuous activities or in specific environments. The increase in victims of heat stroke, even in warm climates, may be attributed to global warming. Heat stroke can lead to multi-organ failure (MOF) and cause myocardial damage.

On the other hand, stress-induced (takotsubo) cardiomyopathy is a transient myocardial dysfunction precipitated by physical or emotional stress, mimicking the presentation of acute coronary syndrome [1].

Here, we report a case of heat stroke presenting with wide QRS tachycardia and shock, the cause of which was considered to be stress-induced cardiomyopathy.

Case report

An 87-year-old man was brought to our emergency department because of consciousness disturbance and generalized convulsion on a hot summer morning. He had a 30-year history of epilepsy and hypertension, which had been treated by a neurologist until 17 months previously, with no subsequent follow-ups and medications. He lived in a house without air conditioning. He was barely able to walk indoors, and had not been eating properly recently. On the morning of admission, his son found him conscious but immobile in the bathroom. His son called an ambulance because the patient gradually became unresponsive and had a convulsion.

On physical examination, he was unconscious (Glasgow coma scale 6 points), his body temperature was 41.2 °C, heart rate 160/min, and blood pressure 110/43 mmHg. Respiration rate was 40/min, and oxygen saturation 96% with O_2 9 L/min mask inhalation. Examination of the chest and abdomen was unremarkable.

Blood examination revealed leukocytosis, and elevated levels of C-reactive protein (1.37 mg/dl: normal value <0.30 mg/dl), aspartate transaminase (82 U/L: normal range <40 U/L), lactate dehydrogenase (444 U/L: normal range <245 U/L), γ-glutamyl transferase (109 U/L: normal range <75 U/L), blood sugar (356 mg/dl: normal range 70–109 mg/dl), and uric acid (12.4 mg/d: normal range <7.0 mg/d). Renal impairment (blood urea nitrogen/creatine = 38/1.88 mg/dl: normal range <22/1.04 mg/dl), and hyponatremia (126 mEq/L: normal range 135–147 mEq/L) were also noted.

A 12-lead electrocardiogram revealed regular wide QRS tachycardia of a rate of 160/min with superior axis and atypical complete right bundle branch block (CRBBB) pattern (Fig. 1). A chest radiograph showed cardiomegaly without pulmonary edema. Echocardiography showed diffuse severe left ventricular hypokinesis.

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http://dx.doi.org/10.1016/j.jccase.2015.03.010
1878-5409/

Fig. 1. 12-lead electrocardiogram at initial presentation. It showed regular wide QRS tachycardia with superior axis and atypical complete right bundle branch block morphology.

As a tentative diagnosis of heat stroke and wide QRS tachycardia due to unknown cause was made, rapid infusion of 2 L cold normal saline and surface cooling with ice pack were started. Intravenous administration of lidocaine (50 mg) and magnesium sulfate (2.46 g) resulted in repetitive transient termination and relapse of tachycardia. Thus, the patient was intubated with sedation by intravenous propofol, which led to gradual slowing of the wide QRS complex tachycardia, and transition to atrial fibrillation (AF). A 12-lead electrocardiogram after wide QRS tachycardia termination showed AF with left anterior hemiblock and CRBBB. These findings, except AF rhythm and slightly different morphology in precordial leads, had been also noted 17 months prior to admission. Acute myocardial infarction was excluded because neither definite ST elevation nor local asynergy in echocardiogram was recognized.

His body temperature returned to 37.5 °C in 4 h by vigorous cooling. Although blood pressure was preserved (100–110 mmHg) during wide QRS tachycardia, he fell into shock (blood pressure 50–60 mmHg) after conversion to AF. Continuous infusion of norepinephrine (0.3 μg/kg/min) to maintain blood pressure above 90 mmHg and heparin to prevent thromboembolism were initiated.

On the next day, blood examination revealed marked increase of transaminase [aspartate transaminase (7521 U/L), alanine transaminase (2636 U/L), and lactate dehydrogenase (5321 U/L)], indicating rhabdomyolysis and/or liver shock. Myoglobinuria (37,600 mg/dl) and elevated creatine phosphokinase (CPK) (4154 U/L: normal range <250 U/L) indicating rhabdomyolysis due to heat stroke and positive troponin-T (1.65 ng/ml: normal range <0.012 ng/ml) indicating myocardial damage were also noted. Thrombocytopenia ($4.4 \times 10^4/\mu l$) accompanied by prolongation of prothrombin time (56.2%: normal range 66.0–127.6%), activated partial thromboplastin time (46.8 s: normal range 25.1–39.8 s), and increased fibrin degradation product (21.3 μg/ml: normal range <5.0 μg/ml) suggested disseminated intravascular coagulation (DIC).

Electrocardiogram on the following day showed giant negative T wave in II, III, aVF, and V2–6 (Fig. 2). Re-examination of echocardiography demonstrated akinesis or severe hypokinesis at mid to apical wall with preserved basal wall motion (Fig. 3). Temporal change of electrocardiogram and echocardiographic findings suggested stress-induced (takotsubo) cardiomyopathy. He was given carvedilol (2.5 mg/day) and enalapril (2.5 mg/day) orally.

There was no recurrence of wide QRS tachycardia and hemodynamics were stable after tapering and cessation of noradrenaline infusion. On the 4th day after admission, he recovered consciousness without evident neurologic deficit and was extubated. One delivery of electrical shock with 100 J for AF on the 5th day restored sinus rhythm, with no recurrence of AF thereafter. On the 7th day, apical wall motion was still hypokinetic but clearly improved. On the 14th day, echocardiogram showed complete recovery of left ventricular wall motion with ejection fraction of 66%. Liver enzyme, CPK, coagulation disorders, and renal function were gradually normalized. Multi-detector computed tomography (MDCT) showed normal coronary tree. His condition continued to improve and he was transferred to another hospital to continue rehabilitation.

Discussion

Heat stroke is an acute thermoregulatory failure due to exposure to high temperatures. Excessive heat denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to diffuse tissue derangement. Complications of heat stroke include acute renal failure, rhabdomyolysis, DIC, acute respiratory distress syndrome, and brain damage. MOF and death will ensue in severe heat stroke.

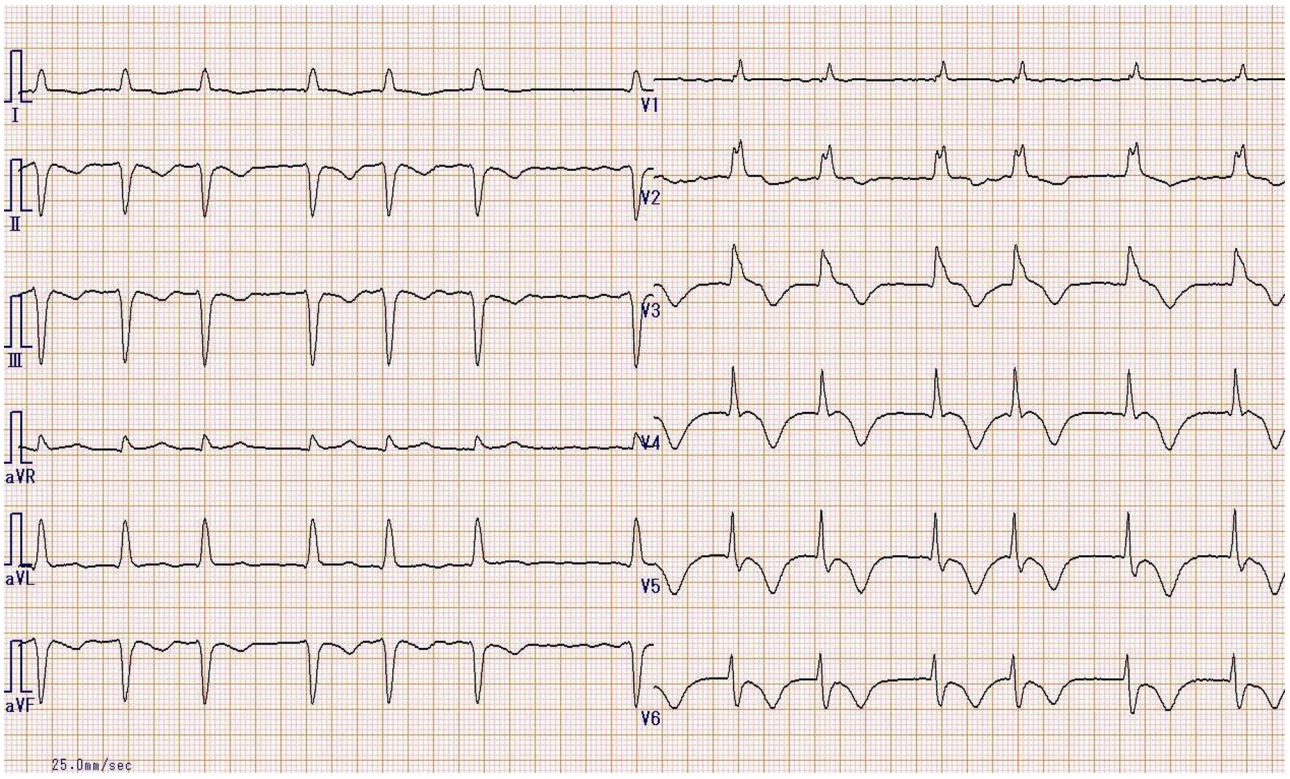

Fig. 2. 12-lead electrocardiogram on the next day. Giant negative T waves in I, II, III, aVF, and V2–6 were newly developed.

Reports about myocardial involvement are scarce and the mechanism of cardiac dysfunction evoked by heat stroke has not been fully understood. Electrocardiographic abnormalities, including regional ST elevation suggestive of acute myocardial infarction, diffuse non-specific ST-T change, conduction defect, prolonged QT interval [2], and such echocardiographic findings as segmental or diffuse wall motion abnormalities and pericardial effusion, have been reported. However, few studies have mentioned stress-induced cardiomyopathy to explain these findings [3,4].

In our case, echocardiogram after termination of wide QRS tachycardia showed circumferential mid to apical severe hypokinesis despite preserved basal wall motion, which could not be explained by myocardial ischemia given the normal coronary anatomy. Absence of regional ST segment elevation or new Q waves in electrocardiogram, complete recovery of wall motion in 2 weeks, and normal coronary arteries proved by MDCT argue against the diagnosis of acute myocardial infarction. Taken together, this case meets the Mayo criteria for the clinical diagnosis of stress-induced (takotsubo) cardiomyopathy [5].

Several mechanisms have been proposed for stress-induced cardiomyopathy, including catecholamine-induced cardiotoxicity, microvascular dysfunction, coronary vasospasm, obstruction of the left ventricular outflow tract, and estrogen deficiency. Production

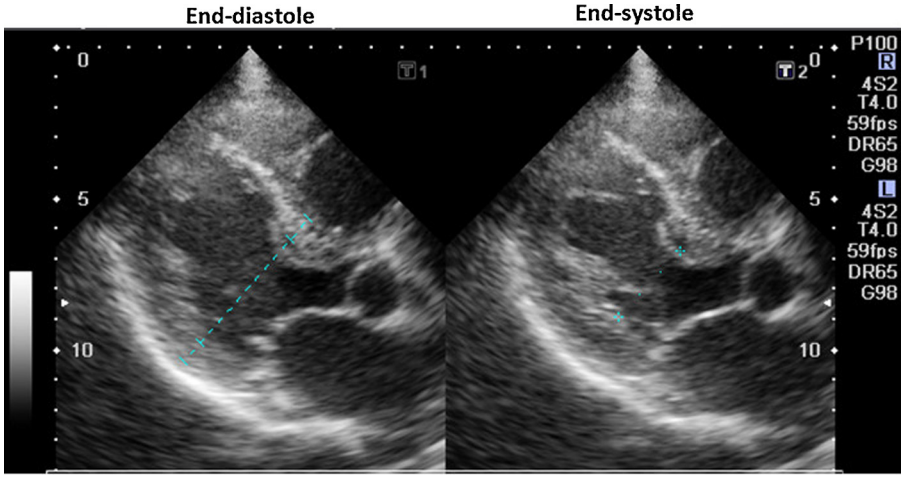

Fig. 3. Ultrasound echocardiogram on the next day. Diffuse wall motion abnormalities at mid to apical wall of the left ventricle despite preserved basal wall motion were demonstrated. (Left) End-diastolic phase. (Right) End-systolic phase.

of cytokine, thrombosis, endothelial cell damage, and increased levels of serum catecholamines [6] (enhanced sympathetic activity), all of which are considered to be related to stress-induced cardiomyopathy, have been demonstrated in heat stroke. In our case, preserved blood pressure despite wide QRS tachycardia and low cardiac output at initial presentation, and paradoxical fall in blood pressure after termination of tachycardia could be explained by increased systemic vascular resistance due to a catecholamine surge induced by heat stroke, followed by rapid decrease of sympathetic activity by therapeutic general anesthesia and cooling, although the serum catecholamine level in the acute phase had not been measured.

Generally considered to have benign prognosis from its transient nature with complete recovery, circulatory collapse necessitating mechanical support or lethal arrhythmia could occur and mortality has been reported in about 2% of stress-induced cardiomyopathy cases. Increased levels of catecholamines in heat stroke could also potentiate the vulnerability to ventricular arrhythmia.

We considered at first that wide QRS complex tachycardia as indicating ventricular tachycardia, because it satisfied Brugada algorithm (R/qR in V1 + rS in V6) [7] and Vereckei aVR algorithm (monophasic R in aVR) [8]. However, these algorithms are inaccurate and make mistakes in every third case, and supraventricular rhythms, especially in serious clinical conditions and/or with depressed left ventricular function, can easily mimic ventricular tachycardia [9]. Careful analysis revealed slightly irregular R-R interval and the comparison with electrocardiogram after conversion into sinus rhythm showed the same axis and similar morphology in precordial leads. Therefore, we retrospectively judged the initial wide QRS complex tachycardia as AF with rapid ventricular response and aberrant conduction. The nature of the transition from wide QRS tachycardia to AF also supports this diagnosis.

Previous studies have demonstrated that hemodynamic status in patients with heat stroke can be divided into two groups: a hyperdynamic group with elevated cardiac index (C.I.), low systemic vascular resistance (SVR), and elevated central venous pressure (CVP), and another hypodynamic group with low C.I., high SVR, and elevated CVP [10]. The first-line therapeutic strategy for heat stroke is immediate cooling and fluid administration. However, rapid excessive fluid repletion may induce pulmonary edema if the patient is complicated by cardiac dysfunction evoked by heat stroke regardless of the underlying mechanisms. To treat a patient with severe heat stroke, special caution, including non-invasive assessment of cardiac function such as echocardiography and electrocardiographic monitoring, and invasive hemodynamic monitoring before the fluid challenge in cases complicated by myocardial involvement, is strongly recommended.

Conflict of interest

None.

Acknowledgments

None.

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